Eleventh Meeting of the Health Effects Advisory Committee (HEAC)
Permissible Exposure Limits for Airborne Contaminants in the Workplace
California Code of Regulations, Title 8, Section 5155
June 4, 2019
Elihu Harris State Building, 1515 Clay Street Oakland, California
Minutes

Division of Occupational Safety & Health
Panel: Garrett Keating, Chris Kirkham and Eric Berg
Notes: Kevin Graulich and Keummi Park

Speaker
Christine Whittaker, Ph.D., Chief, Risk Evaluation Branch, Education and Information Division, NIOSH

HEAC Members Present
Eric N. Brown, Dr PH, CIH, CSP, SCS Engineers (Industrial Hygiene)
Michael N. Cooper, MS, MPH, CIH, Principal Scientist, Mcooperconsulting LLC and UC Davis (Industrial Hygiene)
William Forest, MPH (Epidemiology/Toxicology)
Sarah Janssen, MD, PhD, MPH, Occupational Medicine Department, Kaiser Permanente (Occupational Medicine)
Patrick Owens, MSPH, CIH, Shell Oil Martinez Refinery (Industrial Hygiene)
Kent E. Pinkerton, PhD, UC Davis (Toxicology)
Howard Spielman, Health Sciences Associates and CA Industrial Hygiene Council (Industrial Hygiene)
Mark Stelljes, PhD, SLR International Corporation (Toxicology)
James Unmack, CIH, Unmack Corp (Industrial Hygiene)

Public and Interested Parties Present
Kristin Cummings, California Department of Public Health, HESIS
Mary Deems, California Department of Public Health, HESIS
Stephen Derman, MediShare EHS
Michael Geyer, KERNTEC Engineering
Michael Horowitz
Chris Laszcz-Davis, Occupational Safety & Health Standards Board and NIOSH Board of Scientific Counselors
Dan Leacox
Erika Monterroza, Department of Industrial Relations, Office of the Director
Bob Nocco, Chevron
Natalie Rainer, Keller and Heckman
Maggie Robbins, Worksafe
David Ross, California Department of Transportation
Lauren Scott, American Chemistry Council
James Seward, University of California and Division of Occupational Safety and Health
Kashyap Thakore, California Department of Public Health, HESIS
Elizabeth Treanor, Phylmar Group
Kathleen Vork, OEHHA
Emma Wilson, California Department of Pesticide Regulation

Below are detailed notes of the advisory meeting. These notes do not represent a transcript of the meeting, and are simply a summary of the notes taken by the people conducting the meeting.

Chris Kirkham opened the meeting. The committee members introduced themselves and **Kirkham** introduced the staff and new Chief of HESIS, Kristin Cummings. He also covered housekeeping items and explained the agenda.

NIOSH Chemical Carcinogen Policy

Kirkham introduced Dr. Christine Whittaker, Chief, NIOSH Risk Evaluation Branch, to do a presentation to the committee on the NIOSH Chemical Carcinogen Policy.

Christine Whittaker spoke about the development of the NIOSH Chemical Carcinogen Policy and focused on the rationale and decision process behind establishing 1 excess case of cancer in 10,000 workers exposed for a 45-year working lifetime as the target risk level. See her slides for details.

Mark Stelljes asked about using 45-year working lifetime as a standard industrial worker would not be staying 45 years in the same job. **Whittaker** explained that a part of this was from the tradition when people used to stay with one employer for the working lifetime. Even if they were not exposed from the same employer, they might move to another employer and have similar exposures. **Kirkham** stated that it had been challenged in the federal court for OSHA rulemaking and OSHA prevailed on setting the PELs with the assumption of 45-year working lifetime. **Whittaker** added that there was a language in the OSHA Act that stated no employee should suffer and there certainly were people who worked for 45 years. **Stelljes** stated that it was inconsistent with how worker exposure evolved in EPA where they had used 25 years for occupational exposure assessment since 1989. **Whittaker** stated that EPA was also doing the occupational exposure assessment under TSCA and those assumptions might be changing.

Patrick Owens stated that changing the target risk level from 1 in 1000 to 1 in 10,000 did not necessarily lower the RML-Ca value by a factor of ten. **Whittaker** concurred and stated that it just changed the risk level by ten-fold.

Maggie Robbins, Worksafe, stated that she was puzzled why 1 in 1000 risk level was okay for workers but not for community exposure. **Whittaker** stated that it was one of the issues NIOSH deals with – why accept higher risk for workers than for people in the environment? Whittaker said NIOSH broke down the problem into a series of questions. Why is cancer risk different from other chronic disease and should it be? A: The seriousness and dread associated with cancer warrant a special policy. Are occupational exposures different from environmental exposures? Should workers be more exposed? The benefits conferred from working used to be the justification. The worker chose to work at the jobsite and accepted the risk. The pay was the benefit that offset the risk. The problem was that most people did not know the risks they were exposed to and they did not know that chemicals were carcinogens. **Eric Berg** stated that it was not voluntary when people had to work out of economic necessity.

Stelljes stated that a part of rationale he heard was that there was supposed to be only healthy adults in the workplace, as opposed to infants, elderly, and more susceptible people. **Will Forest** commented that people tended to stay at workforce if they were healthy and not the people who were made unhealthy and driven out of the workforce. **Stelljes** stated that people did not work if they were unhealthy in every field not just the occupation exposed to the chemicals. **Forest** stated that it was not that people were only supposed to work if they were healthy, rather people tended to work if they were healthy. **Stelljes** stated that was how EPA

described in their justification for differentiating between occupational and environmental risks in their guidance document.

Howard Spielman stated that in the past it was assumed that people would know their exposure and people with pre-exiting conditions such as asthma would not be put in those exposures or not hire them. These days HEAC was taking into consideration a certain portion of population that might come into the workforce with pre-existing asthma. HEAC was looking at the standard setting approach to protect people with asthma on the assumption that the employer was not going to keep them out of the exposure. He thinks the NIOSH policy follows that new approach.

Whittaker explained that one rationale for NIOSH changing the risk level was to move away from exposures where workers suffered more risks than people just exposed in the environment. They could not move all the way to 1 in a million risk because that would be a huge jump, but moving in that direction was a part of the rationale.

Whittaker said the next is issue was what language is used to describe the risk level. NIOSH adopted the phrase of target risk. **Whittaker** stated that OSHA looked at a "significant risk" because of the 1980 benzene decision, which stated that a reasonable person would consider 1 in 1000 risk of getting leukemia after exposure to benzene to be significant while 1 in a billion risk of getting cancer would not. She explained that when OSHA used 1 in 1000, it was clear they were reducing significant risk. If the final risk was significant, then anything above it was also significant. So they could clearly show that they were reducing the significant risk and they were often constrained by economic and engineering feasibilities. The idea was that OSHA was reducing **significant** (emphasis added) risk but that did not mean this was necessarily acceptable or the level that workplaces should target.

Whittaker stated that as an agency they were looking at population risks, but as an individual, people were looking at risks to themselves. NIOSH has tried to characterize the individual risk. For example, the individual risk of getting a cancer in lifetime was about 0.32. As an individual, people were not worried about increasing that risk to 0.321 for their job if occupational cancer risk was 1 in 1000 if they worked in the job for 45 years. **Owens** stated that people might not be worried because they would not think they would be working at that job for 45 years. **Whittaker** stated that if you looked at a population with millions of workers, the risk of death was in hundreds and thousands of people. She added that people did not understand the denominator and the risk of 1 in 10000 or 1 in a billion were sometimes considered the same very low number.

Whittaker said the next question for NIOSH was what is the right level? **Whittaker** stated that they strived for reasonableness in determining the target level of risk. **Stelljes** stated that environmental levels were set to protect everyone and the goal was not to have anyone with cancer, whereas occupational levels were protecting a population in the workforce. **Whittaker** disagreed and stated that EPA calculated the risk of 1 in 1000 or 1 in 10,000 depending on the hazard. Workers were a subset of the general population and they tended to be healthier and within a certain age range. She asked what evidence supports a target risk level? She commented that there was no scientific evidence to support a specific target risk level and it was a societal value decision.

Whittaker continued with her presentation to cover the NIOSH history of target risk and target risk levels set by other organizations.

Michael Geyer, KERNTEC Engineering, stated that a lot of RML-CAs were based on the limit of quantifications (LOQ), which depended on the method. He asked whether NIOSH would follow RML-CAs with LOQ and the advancement of technology. **Whittaker** explained that NIOSH did not update anything automatically. Instead

NIOSH would go back and review them individually. She wanted to make it explicit what NIOSH thought the risks were when NIOSH sets an RML-CA higher than the target risk level, such as setting the RML-CA to an LOQ.

Kristin Cummings, CDPH/HESIS, asked if they were going to include a value at 1 in 10,000 risk level even if it was less than LOQ. **Whittaker** replied that they were revising how to organize the NISOH Pocket Guide at the moment and she hoped that information would be included.

Kashyap Thakore, CDPH/HESIS, asked how they evaluated genotoxic versus non-genotoxic carcinogens, single carcinogen versus mixture, incomplete carcinogen versus complete carcinogen and initiators versus promotors. **Whittaker** stated that for genotoxic and non-genotoxic carcinogens, they only had one example of how they looked at them. They concluded that titanium dioxide was carcinogenic by a secondary genotoxic event. They had data for lower doses and higher doses and they used the dose response curve, which was sublinear, to characterize the risk. This was the method they would use if they had that information. But a lot of times they did not have that information and they might have some hints that it might not be genotoxic. However, that was not enough to reject the linear model. They needed to have affirmative reasons to believe that something was not genotoxic. The easiest way would be if there was clear evidence that the mechanism of action was a non-genotoxic mechanism and could justify a nonlinear modeling approach.

Thakore asked how they considered the threshold aspect of non-genotoxic. **Whittaker** stated that for titanium dioxide it was sublinear and that kind of idea got over at the threshold idea. They could still estimate risks because the idea of whether it was a firm threshold and whether below that level was completely safe was not all that clear because of the variability in population and other things. They did not know what that threshold would be that was truly safe. They avoided that issue by looking at the sublinear dose response curve. **Stelljes** stated that EPA did the same sort of thing with chloroform where they were protecting cell hyperplasia, precursor to development of cancer. They were protecting a non-cancer endpoint, which was the required precursor for cancer. But one needed enough data to support that as a mechanism. **Whittaker** added that it was more of a data issue than anything.

Whittaker stated that NIOSH did not deal with mixtures very well yet and they were looking at different ways to deal with them. For complete and incomplete carcinogens, it was completely data driven and it depended on what the data showed.

Chris Laszcz-Davis, OSHSB and NIOSH Board of Scientific Counselors, stated that her approach often was to look at an issue from the operational stand-point and how it translated. She asked what approaches to take and how to accommodate the proliferation of advanced materials, nanomaterials etc. **Whittaker** stated Chuck Geraci had done good work on this and it involved more statistics. It was like a read-across where you had some sentinel chemicals you could use to describe the toxicity and you could bin chemicals into different places based on how you thought they corresponded to the other chemicals. They were doing a lot of work on nanomaterials for example. It was a hard question as emerging hazards could take many shapes and forms and that was a real issue as well.

Laszcz-Davis asked Whittaker's thought on the gig economy. **Whittaker** stated that under EPA TSCA assessment they were looking at conditions of use and how chemicals were used for specific occupations and workplaces. But NIOSH RELs and RML-CAs were set regardless of how they were exposed to chemicals. NIOSH's approach had different level of analysis. They did not consider gig economy in her unit but other people at NIOSH were looking at that issue.

Spielman stated that he didn't think volunteers on a committee such as himself were technically competent to

conduct cancer risk assessment and he saw the NIOSH resources as a significant potential tool for the HEAC to take advantage of and asked how NIOSH determined the priority on what they looked at. If HEAC made a request on some chemicals for NIOSH to look at for their input, how would that stack up on their priority scheme and what kind of input they could expect to get. **Whittaker** explained that it would depend on what was being requested. They spent a lot of time reviewing other agencies' work. If the request was for comments and suggestions about a work product that had been put together, it would be definitely doable. NIOSH does take request from priority stakeholders and this group was such a priority stakeholder. But if the request was to develop something then it might have a long time arc.

Robbins asked Whittaker's opinion on a critique of how adequately EPA looked at the occupational exposures in general when they were doing their assessments, both looking at exposures and interventions. She thought there was a similar issue at state level with the adequacy of reviews and accommodating actual workplace conditions, getting recommendations and exposure assessments. She asked whether the critique was on the point and if there was a way NIOSH was trying to move EPA to have a broader understanding of occupational issues.

Whittaker stated that they had some significant dialogues with EPA on how they characterized exposures and how they characterized occupational risk assessments. She was very involved in TSCA risk assessments because it required occupational risk assessments. NIOSH worked closely with EPA and provided comments and suggestions. EPA had a history of how they did things for environmental risk assessments that did not always translate well into occupational setting. **Stelljes** commented that EPA did not have any legal jurisdiction to handle occupational limits outside the TSCA. They had been doing environmental based risk assessments for workplaces and he did not believe they had a jurisdiction for that. **Whittaker** stated that EPA would argue under the Lautenburg Act, which was passed a couple of years ago. They were under the statutory deadlines to produce the risk assessments and to produce the risk mitigations. **Robbins** added that the idea was to have toxicologists working together regardless of which bucket they came from, environmental versus occupational, to come to shared understanding about what was going on and it was not happening very well at the moment.

Sulfur Dioxide - Discussion

Keating stated that he had done some analysis on the asthma data, which was the basis for the ACGIH TLV- STEL, to extrapolate to more of a worker general population. The current Cal/OSHA PEL is 2 ppm. The ACGIH recommendation is a 0.25 ppm STEL based on volunteer studies in asthmatics exposed to a range of concentrations at different time points and different breathing rates. After these conditions, they measured FEV1 and/or sRaw in subjects. If there was 25% reduction in FEV1 or 100% increase in sRaw, it was considered a clinical response, which they based their standard on. He explained that he had scaled the asthma response/incidence and breathing rates from asthma studies to the population to relate these studies in sensitive subjects to worker exposure at 15 minutes. Keating discussed the slides. SCOEL uses a similar basis for a standard of 0.5 ppm.

Keating explained that he normalized asthma studies by taking experimental conditions to create a variable (integrated exposure) and normalize the results from different studies. Then he scaled asthma response to population response by dividing number of asthmatics in the study by percentage of asthmatics in the population. He used 10% for the calculation as an estimate. **Owens** asked if the breathing rate was measured at the beginning of the exposure and assumed to stay the same during exposure. **Stelljes** asked if it was integrated over time. **Keating** stated that subjects were put on treadmills to get to the breathing rate and then exposed to SO2.

Keating continued to explain that estimated population response ($Population_R$) was dependent on the time interval of the study (5 or 10 minutes). He time-scaled $Population_R$ (15/5, 15/10) to determine population response at 15 minutes. He developed a relationship to estimate $Population_R$ at different concentrations and breathing rate. He used sRaw response for the asthma table because there were more data points than FEV.

Keating clarified that $Population_R$ values in the table on page 4 were represented in percentage. He used the integrated exposure (IE) and population response at 15 minutes for regression and linear fit of data on page 5. He added that % responding was those experiencing a 100% increase in sRaw, which was considered a serious effect in the asthmatics studies. **Stelljes** asked if Keating had done upper and lower confidence limit bands and stated that the correlation did not seem to be too strong. **Keating** replied no. He stated that the coefficient from the linear fit could be used to scale the integrated exposure to tell what the response would be at a fixed concentration (0.25 ppm) and time (15 min).

Forest commented that the exposure time was taken into account in developing the integrated exposure and then time was used again to calculate the population response at 15 minutes. **Keating** stated the integrated exposure values were dependent on the study exposure time (5 0r 10 minutes) and he scaled them to 15 minutes to convert them to a STEL response. **Cooper** concurred with Forest and stated that time was used twice. **Forest** stated that time was already used in calculating the integrated exposure to normalize the data. **Keating** stated population response values were percentage of population responding at different exposure durations. More individuals would respond if exposure duration was longer. **Stelljes** stated that the second row of the table on page 4 showed the population response of 1 when the exposure time doubled. When the exposure time was double at constant concentration, the response did not double. It went from 0.9% to 1%. He added that he did not understand why Keating used another calculation to change the time term when data showed that it was actually not linear. **Keating** explained that the integrated exposure was not normalizing but rather multiplying everything together. **Stelljes** and **Forest** opined that it was redundant and excess adjustment. **Owens** pointed out that the values on the x-axis would be different if time was taken out from the integrated exposure calculation. **Keating** said that he wanted to know the response at 15 minutes. He stated that he could multiply concentration by breathing rate and divide it by time to get an integrated exposure per minute. Then he could multiply by 15. **Cummings** commented that the unit for integrated exposure was concentration x breath because time units would cancel out. **Owens** suggested to use integrated exposure per minute. **Keating** said it would be an acceptable way to scale to 15 minutes and it would be a better way to represent IE.

Keating stated that once the regression coefficient was obtained, it could be used to calculate a population response for different concentrations and fixed breathing rates at 15 minutes. MET (metabolic equivalents) was the ratio of work metabolic rate to the resting metabolic rate. The table on page 7 showed the extensive list of occupations and their mean MET values. Breathing rate could be determined from MET as shown on page 8. These breathing rates and a concentration of 0.25 ppm were used to calculate a population response by integrating the exposure and multiplying that with the regression coefficient. At 0.25 ppm, a population response of 5% exceeded only with very heavy activities. According to CDC data, most occupations had METs of 1-3 and less than ten occupations had METs of 6 and above. He stated he was trying to relate the asthma studies conducted with high breathing rates to occupational breathing and there were plausible occupations where more than 5% of workers would exceed a 100% sRAW increase at 0.25 ppm. At 0.5 ppm, a population response of 5% was exceeded with moderate activities.

Cooper asked if Keating was going to fix the lack of correlation on page 5. **Keating** said Stelljes had suggested the confidence intervals but he was not sure how that improved the correlation. The other adjustment would be to express IE per minute. **Owens** stated that it would be a good way to use all available study data. **Stelljes** stated that there could be outliers and **Cooper** said it would be helpful to know. **Keating** stated that the asthma studies

used intense breathing rates of 40-80 L/min. **Keating** said he questioned the linear relationship because at higher breathing rates, more air passes through the mouth and directly into the lungs. As the breathing rate went up, the response should increase as more sulfur dioxide was entering the lung. He would adjust the regression analysis as suggested but restated that a linear regression might underestimate the response at moderate and high breathing rates.

Stelljes stated that Keating could use benchmark dose software to get the best fit. **Keating** said there were data requirements for benchmark dose modelling and he was not sure if he could get that from these studies. **Stelljes** offered to help if Keating had questions. The benchmark dose software looked at eight different distributions and picked the best fit. **Keating** said he thought IE was nonlinear and a different model may be more appropriate.

Keating stated that sulfur dioxide had been discussed at three HEAC meetings, mainly focusing on the asthma data. EPA Ambient Air Program did an extensive data analysis on sulfur dioxide toxicology and epidemiology and found that the short-term exposure was the only one that with a causal relationship. Some other endpoints were suggestive but the data were not adequate for a causal relationship. Keating would look at some recent animal reproductive studies and summarize them at the next the HEAC meeting.

Forest stated that he figured out his mistake and not to worry about his earlier comment. The graph on page 5 did not reflect all data points as one population response was 30% and the y-axis only went up to 25%. There was an imperfection to fix but he liked Keating's work. **Owens** asked if it was linear and whether 5 minutes exposure would have the same effect as 15 minutes exposure. **Cooper** answered that they were making that assumption. **Forest** opined that the benchmark dose suggestion made sense. He suggested to do another plot without time factor in the integrated exposure to see whether time mattered by just looking at results.

James Seward, University of California and DOSH, stated that by looking at short-term exposure limit he assumed that most of these studies were one time short-term exposures to people. He asked if there was some evidence that sulfur dioxide at low levels could cause cumulative bronchial damage and short-term exposure that went on daily over a week might indeed produce effects on people. It was important to keep a broader perspective on what really went on in the workplace. **Keating** stated that he would look for that in the EPA analysis as they did very comprehensive review of every sulfur dioxide study.

Cooper asked if there was anything done for FEV1. **Keating** stated that he did not use FEV1 data because there were fewer data points. He said he did not know which had more severity - 100% sRaw or 20% FEV1. **Sarah Janssen** stated that 20% FEV was used more often clinically and it was easier to measure and more accurate.

This ends the morning agenda.

LUNCH BREAK

Selection of Priority 1 Substances for HEAC Review

Keating stated that HEAC needed to set a list of chemicals for the committee to review over next two years. The Priority 1 list had been available on the website for the past two months. If the ratio of PEL to TLV was more than 10, it was considered a likely P1 candidate. The list included concerns of other agencies such as ACGIH and NIOSH and factors they had considered in setting their OELs. The national usage data was represented by tonnage per year in the EPA column, and California usage data was shown in the CERS column as number of facilities reporting storage of the material. The factor in the last column referred to the footnote, which was

taken from the HEAC policy and procedures document.

Cooper made an editorial comment on the list to add the year of last PEL revision to the list. **Keating** went over a presentation about key points of chemicals on the list. The list was in alphabetical order and it was not prioritized. See list and slides for details.

Neurological effects were not considered previously for 1-bromopropane. NIOSH and CDPH had put out a hazard alert on neurological effects of 1-bromopropane. The Cal/OSHA PEL is 5 ppm and is based on developmental and reproductive effects.

There had been an IRIS review on carbon tetrachloride and it has a new RfC and new inhalation risk factor. The Cal/OSHA PEL is 2 ppm or 12 mg/m^3. IRIS values shown in the table were environmental values and needed to be scaled to occupational values.

Dicyclopentadiene was selected from the ACGIH 2019 notice of intended changes (NIC) list, which proposed a factor of 10 reduction based on respiratory and eye irritation and CNS effects.

There were many users of diethylene glycol monobutyl ether in California and there is no Cal/OSHA PEL. There is a provisional RfC from a Superfund program and it is quite low. They used a NOEL and applied an uncertainty factor of 1000 to get the RfC value. The number of users were categorized by SIC code and it showed a wide distribution of users. ACGIH review in 2013 was quoted on the list.

DEHP was listed on ACGIH 2019 NIC with a substantial reduction from the current TLV; the proposed lower TLV is based on reproductive and developmental effects. The Cal/OSHA PEL is substantially higher and not based on these effects.

Methanol had a relatively new IRIS review and it had a developmental effect of reduced brain weight in rats. There was substantial monkey data for methanol, which could be used for risk assessment.

Monochloroacetic acid has no Cal/OSHA PEL and a low TLV of 0.5 ppm. There were over one hundred CERS users but they were primarily universities and biotech companies which used very small quantities. It was on CalEPA's hazardous substances list and it was a severe dermal hazard. There was a case report of 10% surface area dermal exposure leading to fatality. Upon skin penetration, it produced a systemic effect of enzyme inhibition.

Phthalic anhydride had a substantial change in OEL and respiratory sensitization is a concern.

PCBTF was recommended previously by HESIS and it is a widely used solvent in many industries. NTP conducted 2-year rat studies with multiple tumor sites and IARC would be reviewing PCBTF in coming years as well. There is no Cal/OSHA PEL.

Titanium dioxide has a NIOSH REL of 0.3 mg/m^3 for ultrafine particles. The Cal/OSHA PEL is 5 mg/m^3 for respirable particles. Ultrafine particles have a diameter of ≤ 100 nm. A NIOSH document references a sampling method and sampling feasibility does not seem to be an issue. It is widely used in paints, coatings, and cosmetics.

James Unmack stated that larger particles were mainly used for paints and ultrafine particles were used in personal hygiene products such as sunscreen. **Owens** stated that NIOSH Method 0600 was a common method used for respirable particles. **Keating** stated that DTSC did a data call on nanotechnology in California about 10

years ago and nine companies reported handling nanoscale materials.

Stelljes stated that he had been working with 1-bromopropane (1-BP) for 19 years. One of his primary clients solely used 1-bromoprorane and he had done toxicology work for them. There was a new ACGIH TLV that went from 10 to 0.1 ppm. Its basis was peripheral neuropathy based on the vibration sense in workers at a Chinese bromopropane factory, which actually did not exist because there was not enough 1-bromopropane. People went to the places where sampling and testing had been done and they were refused entry or data access. They had used 2-bromopropane before using 1-bromopropane and also used several other chemicals that were never listed in the study. The bigger issue was that the key end point was the vibration sense on the big toe using tuning forks and the doctor served as his own control, which was not acceptable. So the whole study was flawed and it should be thrown out. There were only about fifty people in the study. **Forest** stated that neurotoxic effects of 1-bromopropane had been known before 2006. **Stelljes** stated that he was not arguing the effect but he was arguing the level. It was 500 times lower than any other reported peripheral neuropathy levels and it was just not accurate. The lower TLV was based on only one study. The previous TLV of 10 ppm was also based on one study, which was a range-finding developmental study. It found 0.2 g difference in weights of neonates. The study author printed a retraction in a journal article saying that the difference in weight was due to the change in their procedures. They weighed neonates at the end of the day instead of at the beginning of the day. They went back to their historical data and found that the weight gain for a day was 0.2 g, which was exactly the difference in the study. They did a full developmental study with slightly different dose levels and could not reproduce that end point because they were using the right protocol. Both TLV levels were based on one study and they were flawed. None of the other studies came close to those numbers.

Keating stated that there were hazard alerts on 1-BP. **Stelljes** stated that it was based on the dry cleaning study and that 1-BP had been phased out. There was only one manufacturer that had been doing it as dry cleaning solvent and he had spoken to them the previous week. They informed him that there were only six dry cleaners using it in the country and they were not selling 1-BP to any business in California. **Stelljes** said 1-BP was being used only for vapor degreasing. **Keating** stated that he would look into the extent of the studies and other animal studies. **Thakore** commented that there was a NTP study.

Stelljes stated that ACGIH did not use the NTP study to get their TLV. He added that he had just published an article based on a bioassay they did. It specifically looked for mutations which would support a genotoxic mechanism. It conclusively showed that it was not a genotoxic chemical at target organs at the same dose levels used in the NTP study and it used the same strains of mice and rats. It was not a genotoxic carcinogen and it would not fall into the approach in banding. **Whittaker** stated that the mechanism of action was unknown. Just because it did not show those mutations it did not mean it was not genotoxic. **Stelljes** suggested to lower the priority level of 1-bromopropane because it was not really used in California. **Keating** stated that he would just go through the list, take comments, and revise the list if necessary and come back in next meeting.

Owens asked Stelljes if the current Cal/OSHA PEL of 5 ppm was protective and **Stelljes** replied it was in his opinion. **Janssen** stated that it was based on developmental and reproductive effects and not on neurological effects. **Stelljes** stated when he first started working with 1-bromopropane, he did the benchmark dose modeling on 48 different end points. The end point that resulted in the lowest benchmark dose was the reproductive impact impairment in the F1 generation in the two generation definitive test. That had not changed until this new study came out. **Kirkham** mentioned the CERS data had 61 users. **Stelljes** thought they were all vapor degreasing, which were mostly automated and controlled and exposures were negligible for that specific use. **Forest** stated if it was well controlled, it would not be problematic to set a controlling standard.

Cooper asked when the hazard alert came out. **Stelljes** stated that it was 2011 for dry cleaning. It was based on a

claim made against Stelljes' client by one dry cleaner who claimed that using 1-bromopropane as a solvent caused Hodgkin's lymphoma. But the claimant had worked with perchloroethylene for 30 years and used 1-bromopropane for only 6 months before he got Hodgkin's lymphoma. **Whittaker** stated that she thought there was also neurological impacts in the case.

Mary Deems, CDPH/HESIS, stated that HESIS had put out a hazard alert in 2016 and they were in the process of exercising SB193 authority. They got a customer list for 1-bromopropane products and were working with some of companies on how it was being used, focusing on vapor degreasing, coil cleaning, and other electronics cleaning. She suggested not to make a statement that it was not being used in California until they finished looking at quantities reported being sold in California and evaluating how it was being used. She stated that they got a report of more than 450 customers being sold 1-BP in California. Not all of them were huge quantities but some of them were quite large.

Keating stated that an IRIS review for carbon tetrachloride was done in 2010 and it had been on the P2 list for a while. It had multiple endpoints to consider. Committee members questioned how it was being used although CERS data reported 129 users in California. **Kirkham** commented it might be used in university laboratories and **Spielman** stated that it would be in pint size. **Stelljes** commented that the reason on the list for considering carbon tetrachloride is seriousness of hazard. Although fatty changes in the liver could lead to more serious health effects, it was quite different from liver failure or necrosis. **Keating** stated that it came from the OEHHA risk report and there were some comments to use is to that report. OEHHA had looked at Cal/OSHA PELs and compared cancer and non-cancer end points for about 60 chemicals. They reported the cancer risk of greater than one in 1000 and carbon tetrachloride was one of them. He added that the 2010 IRIS report and the existing PEL had a different end point. **Cooper** asked if OEHHA data was available and **Keating** replied that it was presented at the previous HEAC meeting in 2014. **Keating** stated that he would look at CERS data and find out its distribution.

Cooper questioned whether it was worth looking since there was little difference between ACGIH TLV and Cal/OSHA PEL. **Stelljes** stated that PEL would go down significantly if it was cancer based. **Forest** opined that PEL would not go down that much based on E-4 risk level of 17 $\mu g/m^3$. It might go down tenfold and it would be much less reduction than usual for a cancer based regulation. He explained that the risk level would need to be adjusted from environmental lifelong exposure to the workplace exposure and it might be a total factor of 8 or 10 reduction on rough estimate.

Keating stated that cyclopentadiene formed dicyclopentadiene and the standard applied to both substances. He had not looked at end points but it was in the category of substantial change in OEL values with a factor of ten reduction. **Cooper** stated that it had a small number of users in CERS data. **Forest** commented that the EPA reported volume looked high.

Keating stated that diethylene glycol monobutyl ether did not have a Cal/OSHA PEL and the TLV is 10 ppm. **Stelljes** commented that it had a lot of users. **Forest** stated that everyone used it these days and it had low vapor pressure and low toxicity. It was used as a solvent for paints. He added that it was a safer alternative and he would not want to point people away from it. **Owens** asked why the RfC was so low. **Keating** explained that it was a provisional RfC based on subchronic studies at high dose levels. **Stelljes** asked whether OEHHA or HESIS was looking at updating their report. **Thakore** replied he did not have any information. **Keating** added that there was not a lot of data. **Kirkham** asked if IHs had experience sampling for this in the field. **Unmack** stated that you would not find much glycol ether unless they were heated. **Cooper** stated that he had seen it on Tenax tubes often at very low levels when looking for VOCs.

Forest stated that it probably would not make sense to him but it might be worth looking at based on the provisional RfC to see what that was from. **Stelljes** stated that there was a magnitude of difference between those numbers. **Keating** stated that it was a Superfund Program and they used a lot of uncertainties. **Forest** stated that it should be a relatively easy standard to develop because there was enough data to draw conclusions yet there was not so much that it would be burdensome. It was not particularly complicated or difficult data to develop a standard. **Stelljes** stated that it just had to be extrapolated from ingestion data.

Keating stated that DEHP was selected from among the phthalates; while phthalate use is in flux, DEHP is used almost exclusively in the medical devices industry. **Stelljes** commented that there were only 6 users in CERS database. **Keating** stated that reviewed the CERS database looking for thin film users and companies that extruded materials. They did not list phthalates in the Responder CERS database but upon further investigation, they did use them. It was also an ingredient in many plastics that were being extruded. **Janssen** stated that it was also in food manufacturing. **Keating** stated that he had reached out to some medical device manufacturers and they said they used thin films. **Stelljes** stated that medical device manufacturers would not be exposed to inhalation at workplace setting. **Unmack** stated that heating the polymer with solvent might release DEHP.

Thakore stated that OEHHA classified DEHP as a carcinogen and developmental toxicant and IARC and NTP classifications were 2B and 2 respectively. OEHHA also developed an inhalation unit risk factor. **Keating** stated that it (the ACGIH TLV) was based on irritation and possible neurotoxicity and the Cal/OSHA PEL was not based on current toxicology. DEHP was more commonly used among phthalates and there were new ones coming into the market. **Cooper** commented that they were coming out of the market as a plasticizer.

Robbins suggested that it might be worth collaborating with CDPH to get the information on who was actually using DEHP by using SB193 authority to ask vendors about people who were using it. **Keating** stated that HESIS was committing to one chemical at a time and he thought its toxicology warranted the HEAC review. It was an inhalation hazard where it volatilized and worker studies showed reproductive effects in males. **Janssen** supported the review of DEHP.

Cooper asked if DEHP was included in the ongoing CDPH biomonitoring study. **Janssen** stated that it was included in the NHANES although she was not sure about California biomonitoring program.

Michael Horowitz stated that there was a utility to have a PEL for a substance with increasing usage. If some other form of phthalate were to replace DEHP and similar problems were found later, having a PEL for a related chemical could be influential. He did not think the lower usage in California should prevent from setting a standard. If the information was clear, then HEAC should proceed with a review. **Kirkham** stated that the Cal/OSHA PEL was at particulates not otherwise regulated level and it was pretty high.

Deems stated that not everything used in California was represented in CERS database because there was a threshold reporting level and it was for storage. If DEHP was widely used and hazards were great, the fact that there were not a lot of facilities reporting storage over the threshold level did not mean it was not used in California. Based on her experience with SB193 on 1-bromopropane, there were a lot more companies using the chemical than what was reported in CERS database. **Keating** asked if DEHP was on the list for SB193 project. **Deems** replied that it was not the next one but HESIS had a protocol of priorities they had to look at. At this time they were only working on one chemical at a time and she did not think DEHP was high on the list.

Eric Brown opined there was not enough usage data to warrant a HEAC review. It was a huge environmental toxicant but he did not think it was reaching over to the occupational setting to make it to the priority 1 list at this time. There were other substances that were more widely used with known processes or exposures. **Keating**

stated that there was a big summary review of phthalates coming up and would be advantageous to have that. **Janssen** stated that EPA was working on a cumulative risk assessment for a group of phthalates. **Keating** stated he would look into the study and review internally with others given the wide differences of opinion.

Keating stated that methanol has very high usage and EPA revisited its developmental effects in a 2013 IRIS review. There is a lot of pharmacokinetic modeling for methanol. **Kirkham** stated that the ACGIH and Cal/OSHA limits were very similar except for the ceiling limit. **Cooper** asked if it was on the ACGIH NIC list and **Keating** replied it was not. **Cooper** stated that it was not new and other agencies' OELs were at or near the current Cal/OSHA PEL. **Forest** stated that it was on the Prop 65 list for a known reproductive toxicity and they had an inhalation MADL of 47 mg/day. There would be a thoroughly vetted toxicology review on the exact end-point the committee would be looking at and it should not be difficult to know what should be done.

Keating stated there was no Cal/OSHA PEL for monochloroacetic acid and its usage was not extensive. There were cases involving lethal dermal contact where workers were splashed with this chemical. **Forest** opined that it needs to be regulated. Committee members concurred to proceed.

Keating stated that phthalic anhydride was on the P2 list for a while and the committee wanted to look at a sensitizer. It had a substantial change in OEL. **Stelljes** said it should be looked at. **Kirkham** stated that it was presented as one of the OSHA's most interesting health cases at AIHce. A manufacturer was making chemical resistant countertops from a slurry of phthalic anhydride and silica. A high percentage of the workforce had acquired occupational asthma. The exposure was quite high at 15 mg/m^3 phthalic anhydride as 8-hour TWA.

Keating stated that PCBTF had high usage and no Cal/OSHA PEL. **Stelljes** asked whether HEAC should wait for IARC to finish their review, although it would depend on which end point would be used. **Keating** stated that there were an assortment of tumors at different sites. IARC would be reviewing in November 2019 and the report probably would not be out by the next HEAC meeting in December. He thought there were enough data in NTP study to proceed without waiting for IARC review.

Keating stated ultrafine titanium dioxide particles is on the proposed list. **Kirkham** clarified that the 0.3 mg/m^3 in the table was the NIOSH value for ultrafine particles and not ACGIH value. **Spielman** commented that it would be the beginning of studies of nanoparticles at HEAC. **Stelljes** stated that it might be a good one to start with as there was high usage and it would probably go up. **Spielman** said a review of TiO2 toxicity would be based on particle size rather than particle shape. **Unmack** commented that this would be the first one to look at particle count versus particle mass. **Keating** asked if there was any objections and committee members said there was no objections.

Kirkham asked if anyone had experience sampling or analyzing ultrafine titanium dioxide. **Unmack** stated that he had been doing particle count with ultrafine or nanoparticles. **Kent Pinkerton** stated that he had worked with titanium dioxide nanoparticles in experimental studies. There were different forms of titanium dioxide and some were less toxic than others. There were still some controversy as some of things NIOSH found originally had not been reproduced by studies through NIEHS even though the same investigators were involved in both studies. Although the shape was usually not a consideration, titanium dioxide nanobelts were the most toxic of all of the nanomaterials of titanium dioxide. **Spielman** stated that USC had done some studies in the community and engineers had developed an ultrafine particle separator. There were more devices available these days. **Unmack** stated that he had been using direct reading instruments. **Spielman** commented that there was a need for research to pair the particle count and gravimetric together.

Keating summarized substances confirmed for the Priority 1 list – diethylene glycol monobutyl ether, DEHP,

monochloroacetic acid, phthalic anhydride, PCBTF, and titanium dioxide. He would look into MADL for methanol and he would resolve the other ones (1-bromopropane, carbon tetrachloride, dicyclopentadiene). He would come back with answers for questions in December. He would put the list up on the website for stakeholders.

Benzophenone – First Review

Keating briefed the benzophenone summary. Benzophenone is used in paints and cosmetics. It has very few OELs. It has an AIHA WEEL of 0.5 mg/m^3. There is no animal inhalation data. There is a two-generation feeding study in rats (NTP 2006). The developmental studies were important because there may be endocrine effects with benzophenone. It was not a genotoxic but there were tumors in animal studies. It was not an A2G mechanism and there was non-neoplastic kidney damage in rats and mice. The recommendation was 0.25 mg/m^3 for discussion based on the existing health assessments.

Stelljes stated there were only oral studies and the vapor pressure was very low. It might be hard to translate these results into inhalation target level. The first step was to figure out what kind of exposure concentration these ingestion doses could lead to. **Keating** stated there was not much discussion about that. There were a lot of painting scenarios in the REACH database but he could not get off-gassing rates. He would attempt to come back with that information.

Keating stated that Michigan DEQ used benchmark dose modeling on the NTP 2006 rat study. **Stelljes** stated that they assumed 100% inhalation absorption and it did not look like they had accounted for lack of 100% volatilization. **Keating** agreed and continued with the summary. The assessment was based on the cancer risk using the kidney tumor data (due to lack of evidence for the A2G mechanism) and came out with a dose of 0.95 mg/m^3, which was similar to other estimates. The WEEL just applied basic uncertainty factors to a NOEL and the European Food Safety Authority (EFSA) did very extensive modeling. The European study would be a good one to look at and HEAC could adjust absorption factors. EFSA was not a standard authoritative body referenced by HEAC but they used standard benchmark dose modeling and standard uncertainty factors.

Stelljes asked about 47 kg shown in the calculation for the EFSA health assessment on page 19. **Keating** clarified that it was a typo and it should be 0.47 kg.

Keating stated that 4-hydroxybenzophenone, one of the metabolites of benzophenone, was very endocrine active but that there was no evidence of endocrine activity in two generation study with rats. Benzophenone was used in the endocrine assay, it was very weak but a metabolite was estrogenic. In addition, there were 10 different benzophenones using benzophenone is the base model with different functional groups at various locations. BP-2 and BP-4 were implicated much more strongly as being endocrine active. There are standard endocrine assay results and other data to compare different benzophenones. However, there is s not sufficient evidence for the endocrine effect to use as an endpoint as a basis for a PEL.

Keating stated that the usage data was included in the summary. The European agency mainly focused on the painting scenario for dermal absorption. Benzophenone is a very good dermal penetrant and a skin notation was needed. **Kirkham** commented that the WEEL did not have a skin designation. **Keating** stated that WEEL was based on older subchronic studies and benzophenone was used in sunscreen products. He did not think it would be particularly volatile and it might not be a great concern when he came back with exposure information. **Cooper** commented that OARS was in the process of reviewing old WEEL values. **Spielman** asked how section 5155 handled chemicals that were primarily skin absorbers and their potential for inhalation were very minimal. **Kirkham** stated that he was not aware of any chemical on Table AC-1 that only had skin notation without PEL. **Forest** commented that it was the airborne contaminants table. He asked if there was any significant risk of

particulates going airborne as in sprays. **Keating** replied there was risk during paint coating operations. **Forest** said it might not evaporate but it could still be airborne.

Keating asked the committee whether to keep all the data tables in the substance summary. **Brown** suggested to place them at the end of the document and **Cooper** said not to eliminate them.

Turpentine – First Review

Keating briefed the turpentine summary. Turpentine is a mixture of carene and two isomers of pinene. The current Cal/OSHA PEL is 100 ppm and the ACGIH TLV is 20 ppm based on irritation. It is liquid in room temperature and it is a volatile substance. The ACGIH recommendation was based on a series of studies on human subjects exposed to the mixture for 2-hours rather than individual compounds. There was a clear irritation at 80 ppm and the effects at 20 ppm were non-significant. There are many studies of turpentine in sawmills and dust was a contributing factor. Turpentine was a contact sensitizer and there was no notation for contact sensitizer in title 8 regulation.

Unmack and **Forest** noted several typos for carene in the summary document.

Spielman stated that petroleum distillates were used widely as solvents and there was no PEL. He suggested HEAC to consider petroleum distillates. **Kirkham** commented that Stoddard solvent has a PEL of 100 ppm and has a different CAS number. **Owens** stated that pinene was not a molecule commonly seen in the petroleum industry. **Keating** stated that terpenes were plant based.

Unmack asked if some of the biological effects, such as dermatitis and allergies from turpentine, could be from impurities or other compounds present. **Stelljes** stated there was a possibility because they were naturally occurring plant materials. **Keating** stated that dermal results came from case reports and not from people tested for allergic reaction to turpentine. He had not seen classic sensitization studies with guinea pigs or other animals using the constituents of turpentine.

Cooper asked if pure α-pinene could be found. **Stelljes** stated that you might be able to buy a pair of isomers together but you could not separate them easily. **Keating** stated that the ACGIH standard was for turpentine and selected monoterpenes, and that would be the recommendation for HEAC as well. Any of animal toxicology data on a pure compound were mostly on α-pinene, which was 60% of turpentine.
Unmack stated that the other possibility was that since it was derived through a distillation process, it might eliminate some of the other compounds or biologics. **Stelljes** agreed.

Keating reviewed the data shown in the appendix. It showed three volunteer studies which reported a multitude of respiratory measures at 80 ppm. These studies were conducted at 80, 40, and 2 ppm. There was no effect on FEV and there were minor effects on other end points. The effect on sRaw was significant on the last table on page 16. ACGIH did not explain how they derived its 20 ppm recommendation. His guess was they used the concentration x time assumption. These effects were seen at 80 ppm for two hours and 20 ppm for eight hours and would give the same dose.

Keating stated that more evidence for lowering the monoterpenes came from workplace studies. In a controlled study, they put workers with and without filters into a sawmill and exposed them to terpenes. Without the filters, volunteers were exposed to wood dust and monoterpenes and with the filters they were only exposed to the terpenes. Figures 1-2 showed respiratory measures with and without filters. The first figure showed the effect of dust. The second figure showed the effect of terpenes irrespective of dust and it showed a different end

point TLco, which was a measure of gas exchange. It showed the effects of terpenes at the 10 to 20 ppm range.

Brown asked what the toxicological end point was. **Keating** stated that it was interleukin 6, which was a marker of inflammation in lavage fluid, and TLco. **Brown** asked if it would be considered as a material impairment. **Pinkerton** stated that interleukin 6 was an inflammatory cytokine indicating there was a response occurring. It seemed there were effects with or without filters which would suggest that it was not associated with dust. **Janssen** stated that TLco was the diffusion capacity and it showed how well the gas exchanged across the lung. This was more material impairment than interleukin 6 as it showed how well you oxygenate the blood.

Keating stated that there was another study that observed a reduction in diffusion capacity. It came from exposure to sawdust and terpenes. Wood dust is a known carcinogen and its levels have been lowering. He saw this as possibly better support for lowering to 20 ppm than the irritation studies ACGIH had cited.

Keating stated there was little data from animal studies. There was one subchronic pinene study and no animal inhalation study of turpentine. The pinene study was a 2016 NTP subchronic 90-day study with neoplastic and non-neoplastic results. A concentration-dependent increase in epithelium hyperplasia in bladder was found to be rare but it was used as an end point. Lower sperm count with rats and mice were observed in the dose response data. They stated that both end points were potentially of a concern but NTP has cancelled its 2-year chronic study of pinene and he has contacted NTP to clarify.[1] **Forest** stated that even if they cancelled the follow up, these were sensitive and important end points and it should be used for setting a standard. **Stelljes** noted there were a couple of LOELs in the study. **Forest** commented that 100 ppm was the LOEL for the sperm effect. **Keating** stated that he would follow up and focus on the study. He added that no one had done a hazard assessment using these data.

Owens asked if this was the only study published since the ACGIH TLV was updated and whether it used pure α-pinene. **Keating** confirmed and stated that α-pinene was the predominant one at 60%. The PEL would be for turpentine but most animal data would be for a specific terpene. **Stelljes** stated that would be uncertainty as what to do with the other 40%.

Keating stated epidemiological studies had implicated turpentine with some cancers in adults and children. These were all case-control studies of exposure to multiple solvents relying on recall of exposure from years ago. These were not definitive studies but did show elevated odds ratios for turpentine.

Kirkham asked if there was any studies evaluating short-term exposures because he noticed that Washington state and UK had STELs. **Keating** replied he had not identified any. The only basis he found was 2-hour studies with human the subjects, and animal studies. MAK used the systemic effect (kidney) from the pinene study to set their standard at 5 ppm for turpentine.

Stelljes asked if turpentine had a CAS and **Keating** confirmed that it did. **Keating** explained that the ACGIH TLV listed CASs for turpentine and three terpenes. **Owens** stated that if IHs want to analyze for turpentine, they would probably have to ask for all three terpenes and compare the result to turpentine exposure limit. **Stelljes** stated that the relative toxicity of individual compounds was unknown and one might need to assume they were all the same. **Owens** asked how a Cal/OSHA inspector would sample. **Kirkham** replied that they would follow OSHA and NIOSH methods. **Cooper** commented that they probably used the sum of peaks and **Stelljes** agreed.

Keating stated that there was not a lot of usage in the CERS database. Turpentine had been replaced by mineral

[1] CORRECTION: According to a communication from NTP, the NTP alpha-pinene 2 yr study has been conducted and is undergoing review which is expected to take several years

spirits.

Forest commented that epidemiology studies seemed to show consistently elevated cancer risk. There was not a good risk assessment number to derive from that but it was worth keeping in mind.

n-Butanol – corrections to final version

Kirkham reviewed the corrections made to the n-butanol summary presented at the last HEAC meeting. HEAC was proposing a ceiling of 20 ppm based on eye irritation. The other change was to correct the units for sampling from ppm to lpm.

Other Business

Keating stated that a comment from Lawrence Halpern about the NIOSH presentation on cancer policy was received was received on Sunday. **Spielman** said that he (Spielman) was surprised at and did not see the validity of the statement that OSHA had established that it would be technically and/or economically infeasible to achieve the PEL necessary to reduce the risk below 1 in 1000 through engineering and/or administrative controls.

Spielman asked if it would be worthwhile to get NIOSH's input when DOSH had documentation and a recommendation ready to go to the Standards Board. NIOSH policy was the only occupational cancer policy. **Forest** stated that it was just a policy document and HEAC did not have to ask about it. NIOSH was moving in the direction of a more protective 1 in 10,000 risk and that provided the support for HEAC to do the same. It would often be overruled by feasibility issues but they were setting that as a better policy goal. HEAC would calculate the number trying to get there and the NIOSH policy document provided the support. **Spielman** said he would not mind getting an opinion from NIOSH whether they would support the HEAC calculations. **Stelljes** commented it would be more than just straight forward exposure calculation as it would include causal response and types of modeling. **Forest** said that would be fine. **Cooper** stated that he would not mind asking but would not expect to get a timely answer. He would not want to hold up the process. **Spielman** suggested to try one substance and see what kind of response we get.

Cooper stated that there were discrepancies between agencies regarding cancer policy and it would be helpful to see OSHA moving in the same direction with NIOSH. HEAC needed to be careful in saying that was the direction the nation was moving without OSHA weighing in. **Spielman** stated that he understood the sensitivity about it and he saw it as another tool to get more resources into the HEAC process.

Cummings commented that the Netherland group (DECOS, Dutch Expert Committee on Occupational Standards) had been routinely sending their proposal for exposure limits to NIOSH for review throughout the institute. So there was a precedent.

Robbins stated that the issue of 1 in 1000 had always been a problem. It was refreshing to see NIOSH shifting although she was not comfortable that was the proper end point but at least it was moving in the right direction. It was a matter of acknowledging that it was a value judgement and feasibility judgement. It was everything but a health judgement.

Kirkham stated that the court decision did not say one could not be more protective than 1 in 1000. It was just an example of significant risk. **Stelljes** stated that National Contingency Plan had an acceptable risk range of 1 in 10,000 as a starting point and went down to 1 in a million. That ended up being the Superfund process. Most of

the records of decision for Superfund sites for cancer based clean up were 1 in 10,000 and not 1 in a million because of feasibility and other issues. But, 1 in 10,000 was the lowest bar. It was a policy decision and nothing to do with health effects.

Cooper asked if there was a list of substances heading to the Standards Board and offered to help. **Kirkham** stated that formal rulemaking documents needed to be drafted by the staff and he would reach out if help was needed. **Kevin Graulich** stated that TMA, tetrabromomethane, and two other substances (n propanol and cyclohexane) were pending internal review and he was working on 2-butoxyethyl acetate (EGBA) & 2-butoxyethanol (EGBE), which were almost ready for internal submittal. Then there was a next batch of eight substances that included butyl alcohols and acetates, which had just completed HEAC review. **Keating** explained that formal rulemaking document was different from the HEAC substance summary, which was a working document. **Cooper** requested a list of substances and their status in the process for the committee.

Next meeting will be December 3rd, 2019.

Meeting adjourned.